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ORIGINAL PAPER

Critical role of *Helicobacter pylori* in the pattern of gastritis and carditis in residents of an area with high prevalence of gastric cardia cancer

Masoud Sotoudeh · Mohammad H. Derakhshan · Behnoosh Abedi-Ardakani · Mehdi Nouraie · Abass Yazdanbod · Seyyed Mohammad Tavangar · Javad Mikaeli · Shahin Merat · Reza Malekzadeh

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Abstract We have investigated the role of *Helicobacter pylori* infection and of other risk factors of gastritis and carditis in residents of a high-risk area for gastric cardia cancer. During a national population-based endoscopic survey, 508 randomly-selected participants aged ≥40 were enrolled. Mucosal biopsies were obtained from six standard sites. Polymorphonuclear (PMN) and mononuclear (MN) infiltration and combined inflammatory scores (CIS) for chronic gastritis and *H.pylori* were assessed. Relationships of *H.pylori* and reflux esophagitis with these variables were calculated for cardia and non-cardia subsites. Both PMN and MN infiltrations correlated strongly with *H.pylori* infection. For PMN the relationship was maximum for the antrum (odds ratio (OR) = 9.4 (5.2–17.1)) and minimum for the gastric body (OR = 1.7 (1.0–2.9)). There was a significant relationship between carditis and *H.pylori* (OR = 2.8 (1.7–4.9)). A similar relationship was obtained for MN infiltration. In 56% of subjects the mean MN score for the corpus was equal to or greater than that for the antrum. For 59% of subjects the MN score for the cardia was greater than or equal to the antral score. Use of logistic regression revealed that was the main risk factor for gastritis and carditis in all sites. There was an inverse relationship between reflux esophagitis and carditis. *H.pylori* is the main risk factor for gastritis for all sites of the stomach including the cardia; but this relationship is stronger for the antrum and cardia than for the body. Continuous cardia inflammation may contribute to the high incidence of gastric cardia cancer in this region.

Keywords Gastritis · Carditis · *H.pylori* · Gastric cancer

M. Sotoudeh · M. H. Derakhshan · B. Abedi-Ardakani · M. Nouraie · J. Mikaeli · S. Merat · R. Malekzadeh (✉)
Digestive Diseases Research Centre, Medical Sciences/University of Tehran, Shariati Hospital Kargar Shomali Ave, Tehran 14114, Iran
e-mail: malek@ams.ac.ir

A. Yazdanbod
Ardabil University of Medical Science, Ardabil, Iran

S. M. Tavangar
Department of Pathology, Medical Sciences/University of Tehran, Shariati Hospital Kargar, Tehran, Iran

Introduction

Adenocarcinoma of the stomach is the second most lethal cancer worldwide [1]. *Helicobacter pylori*-induced chronic gastritis is capable of predisposing the mucosa of the distal part of the stomach to the development of atrophy, intestinal metaplasia, and adenocarcinoma [2–4]. According to recent estimates, this infection is responsible for 5.5% of all incident cancers as a result of involvement in gastric carcinogenesis [5]. Chronic *H.pylori* infection stimulates the host immune response and causes infiltration of mononuclear (MN) cells into the gastric mucosa and invasion of the glandular and surface epithelium by polymorphonuclear (PMN) leukocytes. This phenomenon causes repeated and ongoing injury and destruction of the epithelial cells [6, 7]. In some patients the lesion progresses to multifocal atrophic gastritis, intestinal metaplasia, and glandular dysplasia, finally ending in adenocarcinoma in a minority of cases [8–10]. Although the effect of other environmental and genetic factors should not be ignored, *H.pylori* is regarded as the first and the most important etiology of this process of carcinogenesis [11]. On the basis of current evidence, *H.pylori*-associated chronic gastritis is the most prevalent concomitant lesion in patients with gastric adenocarcinoma [12].